Mitigating the Effects of Sleep Deprivation on Cognitive Function via Dual Strategy Involving Enhanced Osmosis of Glutamates in Neural Tissue and Duplication of Meningeal Lymph Nodes

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Introduction

As per a 2017 discovery, the brain has its own lymphatic system located in the meninges, a discovery which upended decades of doctrine in the field of neuroscience. Whereas it had previously been thought that the ill effects of sleep deprivation were a byproduct of a failure to consolidate memories, one of our own publications suggested that perhaps a neurotransmitter byproduct was accumulating in neural tissues and inhibiting electrical signals, resulting in decreased function.

In 2022, a researcher in Italy announced that a chemical in the glutamate family had been identified as being the underlying cause of the symptoms associated with sleep deprivation.

With these understandings, not only may educational curricula be updated, but medical interventions to mitigate the effects of sleep deprivation may be developed in order to enhance human function, particularly for modern warfighters.

Abstract

Two primary approaches are called for in order to create a solution for those in situations of sleep deprivation such as troops in war zones. One is a chemical approach aimed at increasing the rate of osmosis of glutamates within the brain (which may have unwanted side-effects) but which would increase the net efficiency of meningeal lymph cells at removing this waste as its rate of circulation may be enhanced by molecules which would cross the blood-brain barrier and could safely increase the rate of diffusion of glutamates when they are generated. This approach has as an advantage not requiring surgical intervention, but has as its disadvantages that it would be difficult to identify chemicals which cross the blood-brain barrier, strongly repel glutamates in order to enhance diffusion and which would not cause harm to the user.

The second of these two approaches is a surgical/bio-medical approach which would increase the total number of meningeal lymph cells through the use of programmed iPS. This approach, although it would require a greater per-user cost, may have a slightly lesser research cost and would be more likely to be side-effect free.

As the duplication of specific cells using an iPS therapy is now well-within our technical capability, provided that a neural performance improvement under conditions of sleep deprivation can be experimentally proven as effective, such an approach would be likely to be safe once perfected.

Conclusion

Beyond applications for mitigating the harmful effects of sleep deprivation, a therapeutic approach which is capable of reducing neural glutamate load would have a distinct nootropic effect, recommending it for use in students and researchers, as well.